Wounds and Canker Diseases on Western Aspen

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Aspen (*Populus tremuloides*) reaches its most magnificent development in the Rocky Mountains, where trees often live to more than 200 years and become forest monarchs. On the better sites it frequently attains heights greater than 100 feet and diameters of more than 2 feet. At its lower elevational limit, aspen is often a scrubby tree found principally along creeks flowing through ponderosa pine forests or shrublands. Still, aspen may grow to timberline (as high as 12,000 feet in elevation) where it takes the form more of a shrub than a tree. Although it is of slight economic importance for wood production throughout most of the West, aspen is appreciated as one of the most esthetically pleasing trees of western mountains, and is a favored habitat of diverse wildlife species.

The bark of western aspen is smooth and white to yellowish or greenish. On very old, large trees it becomes dark and furrowed toward the base. Because the bark is soft and living, the tree is extremely susceptible to damage and subsequent attacks by fungi. Canker diseases are by far the most serious causes of tree mortality. This leaflet is concerned with the trunk wounds and cankers commonly found on aspen in the Western United States.

Trunk Wounds

Wounds are quite common on aspen trunks because of the fragile nature of its bark. The wounds not only cause direct physical damage to the functioning of the tree, but they are also the most common entrance points for canker disease organisms. Furthermore, their recognition is important when diagnosing diseases because the wound scars are easy to confuse with cankers.

Basal wounds, usually the result of low-intensity wildfires, are common in western aspen stands. Falling trees are a common cause of

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other elongate trunk scars. Lightning strikes also cause vertical trunk scars, but usually kill the tree instantly. Lightning also should be held suspect for sudden die-off of aspen groups, especially if lightning scars are present on at least one of the trees. Branches of aspen are frequently broken by wind and heavy early snows. Even branches that die and fall to the ground may leave trunk scars.

The incidence of conspicuous wounds was noted in Colorado, where frost cracks were found on 52 percent, sunscald on 29 percent, and elk damage on 74 percent of sample plots. Although there was no apparent correlation between elk damage and canker diseases in the Colorado study, another study in Rocky Mountain National Park concluded that elk damage increased the number of Cytospora cankers. Also, a recent survey of aspen on an elk range in Wyoming revealed that "barking" wounds occasionally are infection sites for Cenangium canker. Elk "barking" wounds can be identified by the large notches (fig. 1) made by the elk incisor teeth. Moose cause similar wounds. Deer damage apparently is limited to browsing aspen

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Figure 1.—Elk damage. *A*, Recent damage above and old healed wounds below; *B*, tree on right with new and old wounds; left tree with Cenangium canker initiated at elk damage.

sprouts or very small trees. Big game animals also cause rubbing wounds when polishing their antlers.

Probably the smallest mammals that make "barking" wounds on aspen are the voles (fig. 2), and strangely enough they make some of the largest wounds. This type of wound was found on 61 percent of the plots (13 percent of the trees) in the Colorado study. Vole "barking" occurs beneath the snowpack, and is characterized by very small tooth marks on the surface of the bark. While they may chew completely through the bark of sprouts, voles usually remove only the outermost layer of bark on larger trees. This often results in bark desiccation and localized splitting to the cambium, but overall the inner bark remains alive and radial tree growth remains near normal in the "barked" areas.

Birds such as sapsuckers cause local wounds on a few aspen. Sapsucker injury usually appears as checkered drilling high on trunks in or below the crown canopy. Another very common form of injury results from the numerous insects that bore through aspen bark. Aspen borers, especially, cause conspicuous trunk wounds that are probable points for invasion by canker fungi. Other insects invade wounded tissues and enhance their susceptibility to canker fungi.

Unfortunately, people are often

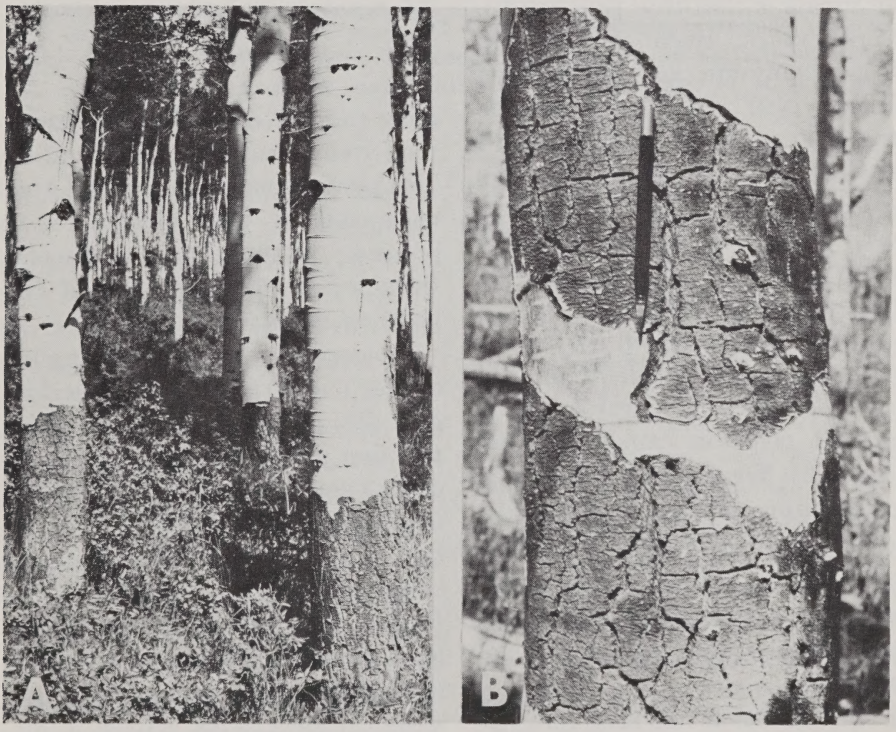

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Figure 2.—Vole damage. *A*, Trees with damage extending upward to 4 feet; *B*, closeup of vole damage to trunk bark.

guilty of causing serious trunk wounds on aspen. Such wounds are frequently made by heavy equipment used to construct roadways, powerline rights-of-way, and the like. Ax and knife wounds are especially frequent in and around campsites. These man-caused wounds often result in localized outbreaks of Cenangium cankers, and thereby initiate deterioration of attractive and useful aspen cover, particularly in recreational areas.

Aspen Cankers

In general there are three types of canker-causing organisms that are recognized by their mode of pathogenic action. (1) **Highly parasitic fungi** penetrate large areas of the bark and cambium, and can girdle a healthy tree within several years. Such is the case with Cenangium and Hypoxylon cankers. (2) **Mildly parasitic fungi** are capable of invading small areas of bark during the tree's dormant period, and then are mostly isolated by callus tissue during cambial activity in the spring. This growth pattern results in cankers characterized by prominent annual callus rings around the origin of infection. Ceratocystis is the most common canker of this group. (3) **Weakly parasitic** and **mostly saprophytic fungi** usually become established only on weakened trees, dying and dead branches, or on damaged bark near wounds. Cytospora canker typifies this group.

Little attention has been paid to aspen cankers in the West because of the tree's limited economic importance. Knowledge about the organisms responsible for canker formation, their mode of infection, or their overall distribution is scanty but increasing. A 1960 survey of aspen in Colorado revealed that cankers were more prevalent than expected, and were generally distributed throughout the major aspen stands. Canker frequency and incidence based on 31 plots in five national forests were as follows:

Canker	Frequency, plot basis	Incidence, living-tree basis
	Percent	*Percent*
Cytospora	97	4. 3
Cenangium	94	2. 4
Ceratocystis	71	4. 1
Hypoxylon	13	0. 2

Nine percent of 4,075 trees examined were dead but still standing. The proportion of these dead trees with cankers was: Cytospora, 54 percent; Cenangium, 51 percent; Ceratocystis, 11 percent; and Hypoxylon, 2 percent.

Cenangium canker.—Cenangium canker (fig. 3) is caused by the fungus *Cenangium singulare*, which was first described in 1888 from decaying aspen bark in Colorado. The organism was later reported on bigtooth aspen in New Hampshire and black cottonwood in Idaho. The fungus was associated with a sooty-bark canker of aspen in Colorado in 1956 and confirmed as the causal agent in 1962. This canker is distributed throughout the range of western aspen from California,

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Figure 3.—The common cankers found on western aspen. *A*, An 11-inch tree girdled by Cenangium canker in 5 years; *B*, a 9-inch tree two-thirds girdled by Hypoxylon canker in 7 years; *C*, a 24-year-old target shaped (below) and 59-year-old (above) black Ceratocystis canker on an 83-year-old tree; D, a 3-year-old Cytospora canker initiated at trunk wound on a 4-inch suppressed tree.

Arizona, and New Mexico northward to Idaho and Washington, into British Columbia. The cankers often get started in older stands that have been subjected to some form of mechanical injury, and once established tend to build up rapidly, even on seemingly unwounded trees. Cenangium canker is one of the major causes of aspen mortality in the West, and is considered the most serious for it tends to occur on the larger trees and on all sites.

The fungus infects trunk wounds and penetrates the inner bark and cambium. Cenangium cankers develop rapidly. Inoculation studies have shown cankers in their first year can extend 40 inches in length and 14 inches in width. In 4 years these cankers were up to 12 feet long by 29 inches in circumference, and had killed some trees.

Young cankers first appear on aspen bark as slightly sunken oval areas with blackened inner bark. The fungus invades bark tissue so rapidly that prominent callus formation is unusual. The bark area killed by the fungus can be seen each succeeding year by the expansion of the original sunken area. The dead outer bark begins to slough off after 2 or 3 years, exposing the blackened inner bark which has become a uniform sooty black. Because the outer bark sloughs off quicker in the central portions, the cankers assume a somewhat concentric zoned pattern. The thick black-

ened inner bark remains tightly attached to the wood for several years, even after the tree dies and falls. It eventually sloughs off in long stringy strips, revealing small black spotted areas on the trunk where fungus mats held the dead bark to the wood. The canker has been termed "sooty-bark canker" because the dead bark crumbles to a sooty-like residue in one's hand.

Apothecia (fruiting bodies) of the fungus (fig. 4) do not usually develop on the recently killed bark, but appear abundantly on the old dead inner bark. The light gray apothecia are about one-eighth inch in diameter, angular to hysteroid in shape, and open when they become wet. The spores formed on the surface are forcibly ejected and wind disseminated when moisture and temperature conditions are favorable. These spores cause new infection if deposited on wounded bark.

Hypoxylon canker.—Hypoxylon canker of aspen (fig. 3) was first found on aspen in the West in the Rocky Mountains in the interior of British Columbia in 1953. Two years later it was reported in Colorado and has since been found in Arizona, New Mexico, and Wyoming. The disease, caused by *Hypoxylon mammatum*, is probably more widely distributed in the West than has been reported. Trees of all ages and sizes on all sites are attacked,

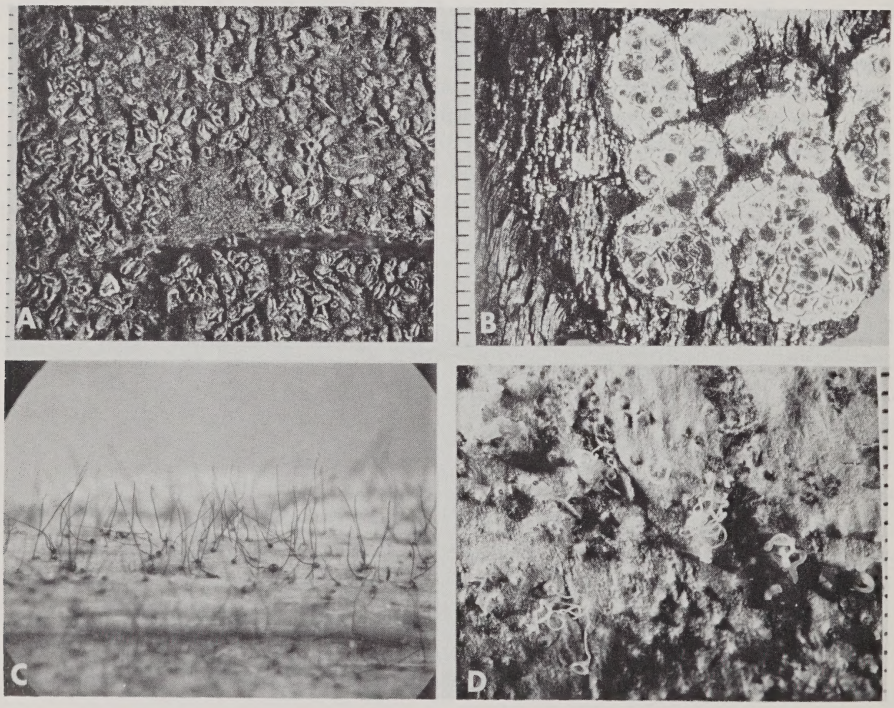

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Figure 4.—Fruiting bodies associated with aspen cankers (scale is mm). *A*, Apothecia of *Cenangium singulare; B*, stroma of *Hypoxylon mammatum* containing embedded perithecia; *C*, perithecia of *Ceratocystis fimbriata* (10X); *D*, spore tendrils of *Cytospora chrysosperma*.

but usually in the more open stands. While the disease causes serious mortality in localized areas, its overall importance remains to be determined.

Because this canker is described in detail in Forest Pest Leaflet 6, it will be treated only briefly here.

Young cankers appear with slightly sunken irregular margins on aspen bark. The inside part of the diseased bark appears laminated or mottled black and yellowish-white. The papery outer bark sloughs off from older infections, exposing a blackened, crumbly inner bark. Canker growth, usually greater in length than width, takes several years or more to girdle a tree.

Small, bristle-like structures are produced under the blistered outer bark about 5 to 14 months after infection. Asexual spores are produced on these structures. Cankers are easier to identify after 3 years, when perithecia (sexual stage fruiting bodies) are formed in small, crust-like stroma up to one-half inch across (fig. 4). Young stroma are covered with a grayish bloom and persist for several years. The faded dead bark in the central portion of the canker then begins to crack in a checkerboard fashion and slough off in small patches.

Ceratocystis canker.—Ceratocystis canker (fig. 3) occurs on aspen throughout the tree's range in the Western United States. Both occasional cankered trees and intensified local outbreaks with many cankered trees are common. It was first described as "black canker" in 1918. *Ceratocystis fimbriata* finally was reported to cause black canker on aspen in Minnesota in 1963. Shortly thereafter, results of studies in Colorado showed that several species of *Ceratocystis* are associated with these cankers, but *C. fimbriata* is the major causal agent. This fungus has a broad spectrum of hosts including sycamore, stone fruits, and coffee.

Ceratocystis canker first appears as a circular, necrotic area on the trunk around a fresh wound or branch junction. During cambial growth in the spring, the tree forms a callus fold at the margins of the canker which temporarily walls off the infection. The fungus invades the new cambium and inner bark during the tree's next dormant season, and kills a new zone of tissue. This process is repeated year after year until a canker, consisting of successive rings of dead bark, is formed. Small young cankers are typically oval or elliptical and appear "target shaped." The dead bark usually adheres to the wood for several years; it then begins to slough off, exposing successive rings of dead woody tissue.

Vertical canker growth is usually greater than horizontal. Because tree circumference growth is generally more rapid than canker enlargement, single cankers seldom kill large trees unless one or more coalesce. The vertical-to-horizontal ratio of canker growth increases with age, and cankers become several feet in length. The fungus is frequently callused off at various places on the canker margin. Older cankers typically have a central area of dead wood surrounded by a

series of bark calluses. These calluses areas may be concentric in outline, but are usually irregular in shape and ragged in appearance due to the massive callus folds and flaring dead bark. The dead bark tissue adjacent to the canker is black; hence the name "black canker."

Black perithecia fruiting bodies of the fungus can often be found with the aid of a hand lens along the canker perimeter in the spring on wood that has been dead for at least 1 year (fig. 4). They are also commonly found on freshly infected trunk wounds. Ascospores ooze out of the long perithecial necks in a sticky mass, and can be accidently picked up by various insects prowling around the canker or infected wound. The disease is thus transmitted by the insects which then visit new wounds. Nitidulids (sap-feeding beetles) are considered the principal vectors.

Cytospora canker.—This disease causes bark necrosis, lesions, and cankers on trunks, large limbs, small branches, and twigs. It is caused by *Valsa sordida*, but the fungus is usually discussed under its imperfect or asexual stage, *Cytospora chrysosperma*. The organism, one of the most common fungi associated with aspen throughout its range, is also common on poplars and numerous other broadleaf trees.

This fungus is a normal inhabitant of the aspen bark microflora, and readily enters and parasitizes bark that has been injured or weakened. The bark area invaded by the fungus may be either fairly regular or very irregular in outline. Small branches and twigs are usually killed without a definite canker being formed. Trunk cankers (fig. 3) are formed by a gradual killing of the bark in more or less circular areas over a period of several years. Annual canker growth can be seen by the slight annual callus formation around the perimeter of the infection, which is usually sunken. The entire bark of large branches and small suppressed trees may be colonized by the fungus in one or two seasons without showing typical canker symptoms. The diseased inner bark rapidly turns dark brown, and the sapwood beneath is stained light brown. The dead bark remains attached to the tree for 2 or 3 years. It then turns lighter in color and falls off in large pieces.

Larger trees may successfully callus off the infection or severely limit canker growth.

When typical canker symptoms fail to develop it is often difficult to recognize the diseased condition until the fungus forms fruiting bodies. These black fruiting bodies or pycnidia of the *Cytospora* stage appear in the outer bark several weeks after infection. Pycnidiospores emerge from the pycnidia in long, coiled, orange to dark red masses called spore tendrils (fig. 4). During rains the spores are partially washed away, leaving sticky masses of spores about the openings; these spores dry to hard hemispherical colored masses. The *Valsa* (sexual) stage of the fungus produces black, flask-shaped perithecia beneath and in a circle around the old pycnidia. Spores issue from the

perithecia and usually collect around the protruding necks in sticky white masses. Both types of fruiting bodies are found on dead bark, but the *Cytospora* pycnidia are more common. Cytospora canker is not considered a vigorous parasite on healthy trees, but certainly hastens the death of trees injured or weakened by other agents.

Control

No chemical control measures are known for these aspen cankers. Removal of cankered trees provides additional space for the residuals, which in turn may become more vigorous and less susceptible to cankers such as Cytospora. Aspen stands should not be opened too quickly, however, because the residuals will suffer from sunscald and the stand might deteriorate rapidly. Also, care must be taken not to wound healthy trees when removing cankered ones. Prevention of man-caused trunk wounds is necessary to avoid a rapid buildup of canker diseases.

If especially high-value trees develop cankers, it sometimes is possible in early stages to excise the causal fungus. All infected bark and wood and the adjacent healthy tissues within 2 inches of the canker margins must be cut away by aseptic techniques, and the newly exposed wood covered with a tree wound dressing.

Since canker diseases frequently increase with stand age, one of the more effective means of minimizing their impact is to manage aspen in small even-aged groups on a short rotation of 80 to 100 years. Silvicultural and harvesting methods that accomplish this will allow aspen to resprout and produce a vigorous new stand. Clearcutting, prescribed burning, and managed wildfires are often effective techniques for regenerating decadent stands. Such treatments may be desirable not only to reduce disease impact, but also to avoid a successional change to less desirable vegetation.

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